**Comment of Dr. Hans Weill, Emeritus Professor of Medicine, Tulane University School of Medicine** (This comment was received after the close of the public comment period.)

I have had the opportunity to read your agency's draft toxicity summary on crystalline silica and the response of the crystalline silica panel. I commend you on a thorough and informative document. I need add nothing to the study-by-study analyses of both your staff and the industry's consultants and agree that limitations always exist in past exposure characterization and ascertainment of disease, but imperfect as they are, it is usually "all we have" and must be subject to the best analyses and interpretations possible.

I would, however, like to make a few comments regarding the clinical reality of silicosis risk in the general population. By way of introduction, I have attached my Resume and CV. As you will note, I have spent most of my academic career in clinical and research activities regarding occupational lung diseases, a major part of this in the study of silica-related diseases. This research has resulted in publications regarding the health effects of sandblasting in the 1970s & 1980s; diatomite mining & milling during the 1990s; sand production in the 2000s; with major reviews & texts on silica-related lung diseases having been published in the 1970s through 1990s – for a total of 30 publications concerning silica-induced disease. The research has been accomplished in collaboration with the members of the multidisciplinary research unit, which I directed for many years, and other US and overseas colleagues. In this letter, I speak only for myself and not for any of these research collaborators, including those engaged in the recent studies of diatomite and sand workers cited in your draft document.

**Comment**. Because of my special interest in the mineral dust diseases, I have personally been asked to clinically evaluate many individuals with actual or putative crystalline silica exposures, both in the workplace and non-occupational environment. I have diagnosed many cases of silicosis, particularly in the decades of the 1960s and 1970s, prominently among sandblasters in the gulf states and periodically in occupational "bystanders" whose work brought them into proximity of abrasive blasting (e.g. pot handlers, painters, welders). I have never seen silicosis in anyone whose silica exposure was to non-occupational sources of crystalline silica. Nor do I believe that the medical and scientific literature provides credible evidence to support a position that risk of silicosis has resulted from silica exposure in the general environment. When downward extrapolations of silicosis risk have been made to crystalline silica levels measured in the non-occupational environment, one must avoid making modeling assumptions (e.g. no threshold, linear relationship) that are not scientifically supportable in assessing the risk of non-malignant respiratory disease in the general population. (We'll leave malignant effects of mineral dust exposures to another day!)

The natural consequence of my views, outlined above, is that I don't believe that an REL for crystalline silica in the general environment is needed to protect the public from a silicosis risk since no such risk has ever been demonstrated.

**Response**. *The chronic REL is explicitly defined as a level at or below which adverse effects in the general population would not be expected. Since the proposed REL is at the upper end of the range seen in the general environment in California, OEHHA is agreeing with the commentator in concluding that silicosis is not likely to be a major problem for background environmental exposures to the general population, but only for those situations where a specific local source may raise exposure significantly above general environmental levels.*

*Current medical practice is unlikely to detect silicosis due to background levels of silica, since (1) most people, except those in dusty occupations, only get chest X-rays when they are ill, (2) the rounded nodules characteristic of silicosis would be few in someone with such putative silicosis due to background silica levels, (3) small irregular opacities, even if due to silica, could not be differentiated from those due to aging or smoking, and (4) autopsy rates in the general population are very low so that such putative silicosis would not likely be detected at autopsy. Hospital autopsy rates have decreased from 41 percent in 1960 to 5 percent in 1997.*

*In regard to a threshold, although regulatory risk assessment for non-cancer effects is moving away from the conventional NOAEL/LOAEL approaches toward a benchmark dose approach, that approach does not necessarily assume the presence or absence of a threshold. In the present case the use of an uncertainty factor to account for inter-individual variation in the general human population (beyond that seen in the specific working populations in the epidemiological studies) implies the assumption of a threshold: there is no process of extrapolation (linear or otherwise) outside the range of observed data in the present calculation.*